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Original Paper

Phantom limb pain

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Abstract

Phantom pain is defined as painful sensation in an absent limb. Many recent studies indicate that 60–80% of amputees experience phantom pain. This research focused on several specific issues related to phantom pain. Specifically, the intensity of pain is similar to those of chronic low back pain, non-terminal cancer pain, and labor pain. The pain is primarily localized in the distal part of the missing limb. Two of the most common descriptors applied to this pain are burning and cramping. The incidence of phantom pain may be greater in males than females and older people may experience this pain more often than younger people. The most recent and most tenable explanation for phantom pain is the new neuromatrix theory defined as a network of neurons that extends throughout widespread areas of the brain, composing the anatomical substrate of the physical self. Some neurosurgical approaches have attempted to reduce the pain, but no long-term success has been reported. Transcuteneous electric nerve stimulation and the administration of pain relief may be an effective treatment.

Key Words: pain, phantom, sensation

Introduction

Phantom phenomena including sensation and pain are serious problems affecting activities of daily living (ADL) for patients with amputation, spinal cord injury, and stroke. In my clinical experience as a physical therapist, I have experienced difficulties treating patients with phantom symptoms, because the problem reduces their motivation to live independently. Many authors (Jensen and Rasmussen, 1994; Ribbers, Mulder, and Rijken, 1989) have found it useful to distinguish between phantom sensation and phantom pain. Phantom sensation is defined as any sensation in the absent limb except pain. On the other hand, phantom pain is defined as painful sensations in the absent limb. Although these are defined as discrete categories, there are many common features across these definitions. In the case of patients recovering from stroke, it may be difficult to tell whether they have a phantom pain. This is because they have not lost their limbs. I investigate phantom limb pain in this paper, because this symptom impairs a patient's ADL more than phantom sensation. Therefore, I have investigated phantom limb pain by focusing on the patients who have undergone limb amputation. This paper outlines research that focuses on several specific issues related to phantom limb pain. Additionally, I refer to the causal explanations of phantom limb pain and treatments.

Prevalence

There is little agreement among researchers concerning the prevalence of phantom pain among patients who have undergone limb amputation. Substantial literature (Abramson and Feibel; 1981; Kolb; 1954; Parkes, 1973) suggests that phantom pain is rare. However, many recent studies (Ehde, Czerniecki, Smith, Cambell, Edwards, Jensen, and Robinson, 2000; Kooijoman, Dijkstra, Geertzen, Elzinga, and van der Schans, 2000; Zuurmond, van der Zande, and Lange, 1996) indicate that 60–80% of amputees experience phantom pain. This lack of agreement has occurred because prevalence rates for phantom pain have been derived from research studies in which the

patient's request for treatment is the only indication of their pain status. Sherman and colleagues (1984) reported that 69% of 2694 military veteran amputees responding to a detailed survey of phantom pain told the researchers that their physicians had directly stated, or clearly implied, that the pain was just in their heads. The great majority of amputees responding to this survey were afraid to tell their physicians that they had phantom pain for fear that the physician would think them insane.

Intensity

Sherman et al. (1984) reported that 51% of 2694 amputees experienced severe phantom pain enough to hinder their lifestyle for more than 6 days per month. Twenty-seven percent of these experienced phantom limb pain for more than 15 hours each day and a further 21% reported daily pain over a 10 to 14 hour period. However, some studies (Melzack and Loeser, 1978; Nikolajsen and Ilkjaer, and Jensen, 2000) report that severe pain occurs in only 0.5 to 5 percent of all amputees. The literature that discusses the intensity of phantom limb pain is difficult to evaluate because it frequently involves case studies that do not describe how phantom limb pain is measured. Dubuisson and Melzack (1976) assessed pain using the McGill Pain Questionnaire and found that phantom limb pain is similar in intensity to chronic low back pain, non-terminal cancer pain, and labor pain. Recent studies (Katz and Melzack, 1987; Marchall and Helme, 1992 ;) that also used this measure reported similar intensities.

Localization

Melzack (1992) and Katz and Melzack (1990) reported the localization of phantom limb pain. The pain is primarily localized in the distal part of the missing limb. In upper limb amputees, phantom limb pain is normally felt in the fingers, palm of the hand, and occasionally the wrist. In lower limb amputees, phantom limb pain is generally experienced in the toes, ball of the foot, instep, top of the foot and ankle.

Quality

Marshall, Helme, and Deathe (1992) and Katz and Melzack (1990) reported on the quality of phantom limb pain. Two of the most common descriptors applied to phantom limb pain are burning and cramping. Other frequently used terms for phantom limb pain are: numb, smarting, stinging, piercing, and tearing.

The Role of Patient Characteristics

In general, studies of phantom limb pain (Ribbers, Mulder, and Rijken, 1999; Jensen and Rasmussen, 1994) suppose that amputees are a homogeneous group. Therefore, little is known about variation within this population. Although some research has examined the relationship between patient characteristics, age, gender, reason for amputation, and levels of phantom limb pain, many yielded mixed results because of differences in sample selection and study methods. Bailey and Moersch (1971) found that the incidence of phantom limb pain is greater in males than females, but another study (Jensen, Krebs, Nielsen, and Rasmussen, 1985) did not identify a difference. Similar difficulties can be found in studies investigating the role of age and medical status. For example, Buchannan and Mandel (1986) found that older persons report the presence of phantom limb pain more often than younger persons; however, Jensen et al. (1985) found no such difference.

Causal Explanations

There have been a plethora of hypotheses advanced to explain the causes of phantom limb pain. Sherman (1989) focused on the role of peripheral nerve fibers in the explanation of phantom limb pain. Following amputation, fibers from the cut end of nerves grow into neuromas, which generate abnormal impulses. These impulses activate central nervous system neurons and may result in the perception of phantom pain. Although this study shows that peripheral factors undoubtedly play a role in phantom limb pain, there is evidence to suggest that this theory does not explain the primary eliciting factor. Pain can also occur in the absence of nerve damage, such as when a limb is congenitally absent, or when information from the periphery is blocked, such as when there has been a complete transaction of the spinal cord (Harwood, Hanumanthu, and Stoudemire, 1992; Melzack, 1992).

Livingston (1983) proposed that phantom

limb pain could be attributed to abnormal firing patterns in the internuncial neurons in the spinal cord. In a study of 36 upper limb amputees suffering from phantom limb pain, he injected a local anesthetic into the sympathetic ganglia in the spinal cord. Although nine of these patients reported a permanent reduction in their pain, more than two-thirds reported a temporary reduction in pain. He suggested that "closed, self-sustaining, reverberating circuits" are set up by chronic peripheral irritation or by the release of spinal cord cells from inhibitory control through the loss of afferent input. When these abnormal impulses reach the brain, they are experienced as pain.

Some researchers proposed that the puzzling aspects of phantom limb pain could be explained by looking at the psychological makeup of the amputee. Parks and Napier (1985) reported that "denier" or "defiant type" amputees had a compulsive need to do everything at least as well as they could before the operation and if possible, better, as if to convince themselves and everyone else that they are not incapacitated at all. They believed that if this defense mechanism were disrupted, for example, by post amputation disability, psychological consequence, such as phantom limb pain would follow.

The latest and most tenable explanation for phantom limb pain is the new neuromatrix theory proposed by Melzack (1992). The neuromatrix is defined as a "network of neurons that extends throughout widespread areas of the brain, composing the anatomical substrate of the physical self". It is suggested that the neuromatrix extends to at least three major neural circuits. One is the sensory pathway that travels through the thalamus to the somatosensory cortex. This pathway is the primary carrier of information from the periphery. A second carrier is the pathway that goes through the reticular formation to the limbic system. This system is critical for emotion and motivation and may account for the affective descriptions of pain used by paraplegics who have complete transaction of the spinal cord and the attendant psychological distress that is commonly seen in patients with phantom limb pain (Coderre, Vaccarino, and Melzack, 1990). The third circuit incorporates the parietal lobe, an area that is significant in evaluating sensory signals and in the recognition of self. The importance of the parietal lobe in the sense of self has been shown in studies of brain-damaged patients. Sacks (1984) reported that patients with damage to the parietal lobe often refuse to accept that their limb is part of them. The limb itself is not damaged, but the patient does not accept it as part of the body. A brain-damaged patient cried out in pain when his leg was strongly pinched but still would not accept that it was his leg that was pinched.

Input to the above systems, either from the periphery or from within the neuromatrix itself is processed simultaneously and then shared with other brain systems, producing output, which is transformed into conscious awareness. Melzack described the basic output from the neuromatrix as a neurosignature, which is particular to the individual. This "neurosignature" may be determined by the pattern and strength of connections between neurons within the neuromatrix. It is this pattern that indicates that the body is intact and unequivocally one's own. In normal circumstances, sensory signals are processed by these systems and modulated by the ongoing pattern of the neuromatrix. The resulting output of the system contains information about the sensory input and the conviction that the sensation is occurring in the body. In many respects, the Neuromatrix Theory is similar to the Gate Control Theory and could be applied to chronic pain in general, rather than phantom limb pain in particular. This theory provides a mechanism to explain how numerous factors can result in the perception of pain.

Treatments

Sometimes surgical treatments for phantom limb pain are attempted. The results have generally been unfavorable (Iacono, Sandy, and Bamford, 1987 ; Malin and Winkelm-Uller, 1985). Various neurosurgical approaches had been used for phantom limb pain, but no long-term successes were reported (Kolb, 1974). Several destructive central nervous system procedures have also been unsuccessful (Kolb, 1974).

Maiorchik, et al. (1980) tried to use electrical stimulation for 14 patients with severe phantom limb pain. They reported that electrical stimulation with a frequency range of 1–20 Hz was most effective, but long-term effects were not observed. I reported the feature of

transcuteneous electric nerve stimulation (TENS) and the difference of the effect of TENS between lower frequencies and higher frequencies (1994). TENS using frequencies less than 20 Hz was used for the long-lasting effect of reducing pain through inhibition of the central nervous system, like morphine. TENS using frequencies more than 80Hz was used for the first and short time effect of reducing pain according to the Gate Control Theory. However, there was a problem of accommodation and both of them are not sufficient for producing long-term effects. Some case reports using electrical stimulation have indicated excellent results whereas others have shown the results to be no better than a placebo effect (Wester, 1897 ; kumar, Nath, and Wyant, 1991).

It was upon the basis of hyperirritable foci that dorsal root entry zone (DREZ) lesions were first attempted. These lesions show significant promise for avulsion injuries and for some other deafferentation syndromes (Young, 1990). However, the long-term results for DREZ procedures for phantom limb pain have been only fair (Saris, Iacono, and Nashold, 1988).

Nikolajsen, Hansen, Nielsen, Keller, Arendt-Nielsen, and Jensen (1996) reported on the use of Mexiletine for central pain involving deafferentation or amputation. The one year follow-up revealed an 87 percent incidence of significant pain relief in a group of 45 phantom limb pain patients. The mechanism of action is unclear, but probably involves the blockage of sodium channels by the mexiletine molecule. This probably occurs in the damaged hyperactive N-methyl D-aspartate (NMDA) receptors and may result in chemical suppression of the hyperirritable foci.

Conclusion

Sixty to eighty percent of amputees experience phantom pain localized to the distal part of the missing limb. The intensity of this pain is similar to chronic low back pain and two of the most common descriptors are burning and cramping. Phantom limb pain may be explained by the role of peripheral nerve fivers, abnormal firing patterns in the internuncial neurons in the spinal cord, the psychological makeup, or the neuromatrix theory, which is defined as a network of neurons that extends throughout widespread areas of the brain, composing the anatomical substrate of the physical self. One method of electrical stimulation, TENS, and the use of pain relief, Mexiletine, may be an effective treatment for phantom limb pain.

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